```
      (FILE 'HOME' ENTERED AT 12:30:13 ON 17 OCT 2002)

      FILE 'BIOSIS, CABA, CAPLUS, EMBASE, LIFESCI, MEDLINE, SCISEARCH,
      USPATFULL, JAPIO' ENTERED AT 12:30:29 ON 17 OCT 2002
L1         17527 S ACTINOBACILLUS
L2        141614 S SUPEROXIDE DISMUTASE
L3           840 S L2 AND VACCINE
L4           534 S L3 AND (ANTIBODY OR ANTIBODIES)
L5             0 S L3 AND (ANTI-ANTIBODIES)
L6           491 DUP REM L4 (43 DUPLICATES REMOVED)
L7             0 S L4 AND PLEUROPNEMONIAE
L8             6 S L4 AND PLEUROPNEUMONIAE
L9             6 DUP REM L8 (0 DUPLICATES REMOVED)
L10        83584 S HAEMOPHILUS
L11          268 S L2 AND L10
L12           65 S L11 AND (VACCINAT? OR INJECT? OR IMMUNIZ?)
L13           65 DUP REM L12 (0 DUPLICATES REMOVED)

      FILE 'STNGUIDE' ENTERED AT 12:41:03 ON 17 OCT 2002

      FILE 'BIOSIS, CABA, CAPLUS, EMBASE, LIFESCI, MEDLINE, SCISEARCH,
      USPATFULL' ENTERED AT 12:43:41 ON 17 OCT 2002
L14          158 DUP REM L11 (110 DUPLICATES REMOVED)
L15           86 S L14 AND (INJECT? OR IMMUNIZ? OR VACCIN?)
L16           68 S L15 AND ANTIBODIES
L17           68 DUP REM L16 (0 DUPLICATES REMOVED)
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L10 ANSWER 1 OF 27 USPATFULL

AB In order to obtain a novel binding protein against a chosen target, **DNA** molecules, each encoding a protein comprising one of a family of similar potential binding domains and a structural signal calling for the display of the protein on the outer surface of a chosen bacterial cell, bacterial spore or phage (genetic package) are introduced into a genetic package. The protein is expressed and the potential binding domain is displayed on the outer surface of the package. The cells or viruses bearing the binding domains which recognize the target molecule are isolated and amplified. The successful binding domains are then characterized. One or more of these successful binding domains is used as a model for the design of a new family of potential binding domains, and the process is repeated until a novel binding domain having a desired affinity for the target molecule is obtained. In one embodiment, the first family of potential binding domains is related to bovine pancreatic trypsin inhibitor, the genetic package is M13 phage, and the protein includes the outer surface transport signal of the M13 gene III protein.

AN 2002:272761 USPATFULL

TI Directed evolution of novel binding proteins

IN Ladner, Robert Charles, Ijamsville, MD, UNITED STATES
Guterman, Sonia Kosow, Belmont, MA, UNITED STATES
Roberts, Bruce Lindsay, Milford, MA, UNITED STATES
Markland, William, Milford, MA, UNITED STATES
Ley, Arthur Charles, Newton, MA, UNITED STATES
Kent, Rachel Baribault, Boxborough, MA, UNITED STATES

PI US 2002150881 A1 20021017

AI US 2001-781988 A1 20010214 (9)

RLI Continuation of Ser. No. US 1998-192067, filed on 16 Nov 1998, ABANDONED Continuation of Ser. No. US 1995-415922, filed on 3 Apr 1995, PATENTED Continuation of Ser. No. US 1993-9319, filed on 26 Jan 1993, PATENTED Division of Ser. No. US 1991-664989, filed on 1 Mar 1991, PATENTED Continuation-in-part of Ser. No. US 1990-487063, filed on 2 Mar 1990, ABANDONED Continuation-in-part of Ser. No. US 1988-240160, filed on 2 Sep 1988, ABANDONED

PRAI WO 1989-US3731 19890901

DT Utility

FS APPLICATION

LREP BROWDY AND NEIMARK, P.L.L.C., 624 Ninth Street, N.W., Washington, DC, 20001

CLMN Number of Claims: 18

ECL Exemplary Claim: 1

DRWN 16 Drawing Page(s)

LN.CNT 15696

L10 ANSWER 2 OF 27 USPATFULL

AB The present invention relates to novel members of the Tumor Necrosis Factor family of receptors. The invention provides isolated **nucleic acid molecules** encoding human TR11, TR11SV1, and TR11SV2 receptors. TR11, TR11SV1, and TR11SV2 polypeptides are also provided, as are vectors, host cells and recombinant methods for producing the same. The invention further relates to screening methods for identifying agonists and antagonists of TR11, TR11SV1, and TR11SV2 receptor activity. The present invention further relates to antibodies that specifically bind TR11, TR11SV1, and/or TR11SV2. Also provided are diagnostic methods for detecting disease states related to the aberrant expression of TR11, TR11SV1, and TR11SV2 receptors. Further provided are therapeutic methods for treating disease states related to aberrant proliferation and differentiation of cells which express the TR11, TR11SV1, and TR11SV2 receptors.

AN 2002:185613 USPATFULL

TI Human tumor, necrosis factor receptor-like proteins TR11, TR11SV1 and TR11SV2

IN Ni, Jian, Germantown, MD, UNITED STATES

Ruben, Steven M., Olney, MD, UNITED STATES
PA Human Genome Sciences, Inc., Rockville, MD (U.S. corporation)
PI US 2002098525 A1 20020725
AI US 2001-915593 A1 20010727 (9)
RLI Continuation-in-part of Ser. No. US 2000-512363, filed on 23 Feb 2000, PENDING Continuation-in-part of Ser. No. US 1998-176200, filed on 21 Oct 1998, PENDING
PRAI US 2000-221577P 20000728 (60)
US 1999-144076P 19990716 (60)
US 1999-134172P 19990513 (60)
US 1999-121648P 19990224 (60)
US 1997-63212P 19971021 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN 11 Drawing Page(s)
LN.CNT 12618
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 3 OF 27 USPATFULL
AB The present invention relates to novel pancreatic related polynucleotides, the polypeptides encoded by these polynucleotides herein collectively referred to as "pancreatic antigens," and antibodies that immunospecifically bind these polypeptides, and the use of such pancreatic polynucleotides, antigens, and antibodies for detecting, treating, preventing and/or prognosing disorders of the pancreas, including, but not limited to, the presence of pancreatic cancer and pancreatic cancer metastases. More specifically, isolated pancreatic **nucleic acid molecules** are provided encoding novel pancreatic polypeptides. Novel pancreatic polypeptides and antibodies that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human pancreatic polynucleotides, polypeptides, and/or antibodies. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the pancreas, including pancreatic cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The invention further relates to methods and/or compositions for inhibiting or promoting the production and/or function of the polypeptides of the invention.
AN 2002:157060 USPATFULL
TI Nucleic acids, proteins and antibodies
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES
Ruben, Steven M., Olney, MD, UNITED STATES
PI US 2002081659 A1 20020627
AI US 2001-925297 A1 20010810 (9)
RLI Continuation-in-part of Ser. No. WO 2000-US5989, filed on 8 Mar 2000, UNKNOWN
PRAI US 1999-124270P 19990312 (60)
DT Utility
FS APPLICATION
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850
CLMN Number of Claims: 23
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 20326
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 4 OF 27 USPATFULL
AB There can be provided a fungal antigen which is an insoluble fraction obtainable from fungal cells of which cell wall has been substantially

removed or at least partially removed; a process for producing the same; a nucleic acid encoding the fungal antigen; a biologic product containing the fungal antigen; a method of stimulating immunological responses by using the biologic product; a method of suppressing allergic reaction to fungi in a vertebrate; and a method for diagnosing a disease caused by fungi in a vertebrate.

AN 2002:112558 USPATFULL
TI Fungal antigens and process for producing the same
IN Takesako, Kazutoh, Otsu-shi, JAPAN
Mizutani, Shigetoshi, Gamo-gun, JAPAN
Endo, Masahiro, Kusatsu-shi, JAPAN
Kato, Ikunoshin, Uji-shi, JAPAN
PA TAKARA SHUZO CO., LTD, Kyoto, JAPAN (non-U.S. corporation)
PI US 2002058293 A1 20020516
AI US 2001-987190 A1 20011113 (9)
RLI Division of Ser. No. US 1999-262856, filed on 4 Mar 1999, PENDING
PRAI WO 1997-JP3041 19970829
JP 1996-255400 19960904
JP 1997-99775 19970331
DT Utility
FS APPLICATION
LREP BIRCH STEWART KOLASCH & BIRCH, PO BOX 747, FALLS CHURCH, VA, 22040-0747
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN 9 Drawing Page(s)
LN.CNT 3093
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 5 OF 27 USPATFULL
AB The present invention provides conjugate compounds comprising at least one heat shock protein or portion thereof including at least one immunostimulatory domain and at least one capsular oligosaccharide or polysaccharide of a pathogenic bacteria. The compound comprises oligosaccharides of the Meningococci C (MenC) group and a heat shock protein selected from M. bovis BCG GroE1-type 65 kDa hsp (hspR65), recombinant M. tuberculosis DnaK-type 70 kDa hsp (hspR70) and a heat shock protein from H. pylori. The invention also provides processes for producing conjugate compounds, pharmaceutical compositions comprising conjugate compounds, therapeutic compositions comprising conjugate compounds, and methods of inducing an immune response.

AN 2002:136574 USPATFULL
TI Conjugates formed from heat shock proteins and oligo-or polysaccharides
IN Rappuoli, Rino, Quercegrossa, ITALY
Costantino, Paolo, Colle d'Elsa, ITALY
Viti, Stefano, Sovicille, ITALY
Norelli, Francesco, Siena, ITALY
PA Chiron S.p.A., Siena, ITALY (non-U.S. corporation)
PI US 6403099 B1 20020611
WO 9317712 19930916
AI US 1994-256847 19941101 (8)
WO 1993-EP516 19930308
19941101 PCT 371 date
PRAI IT 1992-FI58 19920306
DT Utility
FS GRANTED
EXNAM Primary Examiner: Smith, Lynette R. F.; Assistant Examiner: Portner, Ginny Allen
LREP Attwell, Gwilym J.O., Harbin, Alisa A., Blackburn, Robert P.
CLMN Number of Claims: 11
ECL Exemplary Claim: 1
DRWN 8 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1809
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 6 OF 27 USPATFULL
AB The entire genome of pathogenic E. coli strain O157:H7 has been sequenced. All of the genomic **DNA** sequences present in O157 and absent in the previously sequenced laboratory strain K12 are presented here.
AN 2002:70106 USPATFULL
TI Sequences of E. coli O157
IN Blattner, Frederick R., Madison, WI, United States
Burland, Valerie, Cross Plains, WI, United States
Perna, Nicole T., Madison, WI, United States
Plunkett, Guy, Madison, WI, United States
Welch, Rod, Madison, WI, United States
PA Wisconsin Alumni Research Foundation, Madison, WI, United States (U.S. corporation)
PI US 6365723 B1 20020402
AI US 1999-453702 19991203 (9)
DT Utility
FS GRANTED
EXNAM Primary Examiner: Fredman, Jeffrey
LREP Quarles & Brady LLP
CLMN Number of Claims: 2
ECL Exemplary Claim: 1
DRWN 0 Drawing Figure(s); 0 Drawing Page(s)
LN.CNT 1583
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 7 OF 27 USPATFULL
AB The present invention provides the sequencing of the entire genome of Haemophilus influenzae Rd, SEQ ID NO: 1. The present invention further provides the sequence information stored on computer readable media, and computer-based systems and methods which facilitate its use. In addition to the entire genomic sequence, the present invention identifies over 1700 protein encoding fragments of the genome and identifies, by position relative to a unique Not I restriction endonuclease site, any regulatory elements which modulate the expression of the protein encoding fragments of the Haemophilus genome.
AN 2002:50802 USPATFULL
TI Computer readable genomic sequence of Haemophilus influenzae Rd, fragments thereof, and uses thereof
IN Fleischmann, Robert D., Gaithersburg, MD, United States
Adams, Mark D., N. Potomac, MD, United States
White, Owen, Gaithersburg, MD, United States
Smith, Hamilton O., Towson, MD, United States
Venter, J. Craig, Potomac, MD, United States
PA Human Genome Sciences, Inc., Rockville, MD, United States (U.S. corporation)
PI US 6355450 B1 20020312
AI US 1995-476102 19950607 (8)
RLI Continuation-in-part of Ser. No. US 1995-426787, filed on 21 Apr 1995, now abandoned
DT Utility
FS GRANTED
EXNAM Primary Examiner: Campell, Bruce R.
CLMN Number of Claims: 88
ECL Exemplary Claim: 1
DRWN 47 Drawing Figure(s); 47 Drawing Page(s)
LN.CNT 4666
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 8 OF 27 CAPLUS COPYRIGHT 2002 ACS
AB The invention relates to the identification and isolation of a novel sigma 54 (.sigma.54) transcription factor from Vibrio harveyi. The invention further relates to the identification of .sigma.54 interactions with LuxO. More particularly, the invention provides methods for identifying compds.

that regulate bacterial cell growth and virulence by regulating LuxO-.sigma.54 activities.

AN 2001:833360 CAPLUS
DN 135:369158
TI LuxO-.sigma.-54 interactions in Vibrio harveyi and their uses in regulating bacterial cell growth and virulence
IN Bassler, Bonnie L.; Lilley, Brendan N.
PA Princeton University, USA
SO PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2001085773	A2	20011115	WO 2001-US15364	20010510
WO 2001085773	A3	20020808		

W: AE, AG, AL, AM, AT, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, CZ, DE, DE, DK, DK, DM, DZ, EE, EE, ES, FI, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRAI US 2000-202999P P 20000510

L10 ANSWER 9 OF 27 USPATFULL
AB There can be provided a fungal antigen which is an insoluble fraction obtainable from fungal cells of which cell wall has been substantially removed or at least partially removed; a process for producing the same; a nucleic acid encoding the fungal antigen; a biologic product containing the fungal antigen; a method of stimulating immunological responses by using the biologic product; a method of suppressing allergic reaction to fungi in a vertebrate; and a method for diagnosing a disease caused by fungi in a vertebrate.
AN 2001:235097 USPATFULL
TI Fungal antigens and process for producing the same
IN Takesako, Kazutoh, Otsu, Japan
Mizutani, Shigetoshi, Gamo-gun, Japan
Endo, Masahiro, Kusatsu, Japan
Kato, Ikunoshin, Uji, Japan
PA Takara Shuzo Co., Ltd., Kyoto, Japan (non-U.S. corporation)
PI US 6333164 B1 20011225
AI US 1999-262856 19990304 (9)
RLI Continuation-in-part of Ser. No. WO 1997-JP3041, filed on 29 Aug 1997
PRAI JP 1996-255400 19960904
JP 1997-99775 19970331
DT Utility
FS GRANTED
EXNAM Primary Examiner: Smith, Lynette R. F.; Assistant Examiner: Baskar, Padma
LREP Birch, Stewart, Kolasch & Birch, LLP
CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN 9 Drawing Figure(s); 9 Drawing Page(s)
LN.CNT 2782
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 10 OF 27 USPATFULL
AB The present invention relates to peptides which exhibit antifusogenic and antiviral activities. The peptides of the invention consist of a 16

to 39 amino acid region of a human respiratory syncytial virus protein. These regions were identified through computer algorithms capable of recognizing the ALLMOTI5, 107x178x4, or PLZIP amino acid motifs. These motifs are associated with the antifusogenic and antiviral activities of the claimed peptides.

AN 2001:67794 USPATFULL
TI Human respiratory syncytial virus peptides with antifusogenic and antiviral activities
IN Barney, Shawn O'Lin, Cary, NC, United States
Lambert, Dennis Michael, Cary, NC, United States
Petteway, Stephen Robert, Cary, NC, United States
PA Trimeris, Inc., Durham, NC, United States (U.S. corporation)
PI US 6228983 B1 20010508
AI US 1995-485264 19950607 (8)
RLI Division of Ser. No. US 1995-470896, filed on 6 Jun 1995
Continuation-in-part of Ser. No. US 1994-360107, filed on 20 Dec 1994
Continuation-in-part of Ser. No. US 1994-255208, filed on 7 Jun 1994
Continuation-in-part of Ser. No. US 1993-73028, filed on 7 Jun 1993, now patented, Pat. No. US 5464933
DT Utility
FS Granted
EXNAM Primary Examiner: Scheiner, Laurie; Assistant Examiner: Parkin, Jeffrey S.
LREP Pennie & Edmonds LLP
CLMN Number of Claims: 62
ECL Exemplary Claim: 1
DRWN 84 Drawing Figure(s); 83 Drawing Page(s)
LN.CNT 32166
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 11 OF 27 USPATFULL
AB The invention provides novel genes and proteins of Legionela pneumophila. The invention also provides methods of detecting or quantitating L. pneumophila using these genes, mRNAs encoded by the genes, or proteins encoded by the genes as targets. Nucleic acids designed to hybridize with the genes or mRNAs encoded by the genes, or antibodies that bind specifically to the proteins, are used in the methods, and the nucleic acids and antibodies can be provided in kits.
AN 1999:92500 USPATFULL
TI Method and materials for detecting Legionella pneumophila
IN Cianciotto, Nicholas P., Evanston, IL, United States
Hickey, Erin K., Evanston, IL, United States
PA Northwestern University, Evanston, IL, United States (U.S. corporation)
PI US 5935782 19990810
AI US 1996-766858 19961213 (8)
PRAI US 1996-11545P 19960213 (60)
DT Utility
FS Granted
EXNAM Primary Examiner: Myers, Carla J.
LREP Sheridan Ross P.C.
CLMN Number of Claims: 14
ECL Exemplary Claim: 3
DRWN 23 Drawing Figure(s); 18 Drawing Page(s)
LN.CNT 2418
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 12 OF 27 CAPLUS COPYRIGHT 2002 ACS
AB Methods are provided for protecting the eye from degenerative eye conditions by administering prophylactic histidine compns. Also provided are for treating ocular inflammation resulting from various causative agents, by administering therapeutic histidine compns. Further provided are histidine compns. for carrying out the methods.
AN 1998:618371 CAPLUS
DN 129:255004

TI Prophylactic and therapeutic methods for ocular degenerative diseases and inflammations, and histidine compositions therefor
IN Thomas, Peter G.
PA Cytos Pharmaceuticals LLC, USA
SO U.S., 10 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 5811446	A	19980922	US 1997-839805	19970418
	WO 9847366	A1	19981029	WO 1998-US7319	19980417
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
	AU 9873583	A1	19981113	AU 1998-73583	19980417
PRAI	US 1997-839805		19970418		
	WO 1998-US7319		19980417		

L10 ANSWER 13 OF 27 USPATFULL
AB In order to obtain a novel binding protein against a chosen target, **DNA** molecules, each encoding a protein comprising one of a family of similar potential binding domains and a structural signal calling for the display of the protein on the outer surface of a chosen bacterial cell, bacterial spore or phage (genetic package) are introduced into a genetic package. The protein is expressed and the potential binding domain is displayed on the outer surface of the package. The cells or viruses bearing the binding domains which recognize the target molecule are isolated and amplified. The successful binding domains are then characterized. One or more of these successful binding domains is used as a model for the design of a new family of potential binding domains, and the process is repeated until a novel binding domain having a desired affinity for the target molecule is obtained. In one embodiment, the first family of potential binding domains is related to bovine pancreatic trypsin inhibitor, the genetic package is M13 phage, and the protein includes the outer surface transport signal of the M13 gene III protein.
AN 1998:143904 USPATFULL
TI Directed evolution of novel binding proteins
IN Ladner, Robert Charles, Ijamsville, MD, United States
Gutterman, Sonia Kosow, Belmont, MA, United States
Roberts, Bruce Lindsay, Milford, MA, United States
Markland, William, Milford, MA, United States
Ley, Arthur Charles, Newton, MA, United States
Kent, Rachel Baribault, Boxborough, MA, United States
PA Dyax, Corp., Cambridge, MA, United States (U.S. corporation)
PI US 5837500 19981117
AI US 1995-415922 19950403 (8)
RLI Continuation of Ser. No. US 1993-9319, filed on 26 Jan 1993, now patented, Pat. No. US 5403484 which is a division of Ser. No. US 1991-664989, filed on 1 Mar 1991, now patented, Pat. No. US 5223409 which is a continuation-in-part of Ser. No. US 1990-487063, filed on 2 Mar 1990, now abandoned which is a continuation-in-part of Ser. No. US 1988-240160, filed on 2 Sep 1988, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ulm, John
LREP Cooper, Iver P.

CLMN Number of Claims: 43
ECL Exemplary Claim: 1
DRWN 16 Drawing Figure(s); 16 Drawing Page(s)
LN.CNT 15973
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 14 OF 27 USPATFULL
AB The present invention provides a novel protein of pathogenic forms of **Neisseria**, as well as genes which encode PilC, i.e., the pilC loci. **DNA** sequences of pilC genes are useful as probes to diagnose the presence of microorganisms containing type 4 pilin as well as permitting production of polypeptides which are in turn useful in diagnostic tests and/or as components of vaccines. The invention also provides antibodies directed against pilC epitopes. These antibodies are useful for diagnostic tests as well as therapy.
AN 1998:139022 USPATFULL
TI Polypeptides and antibodies useful for the diagnosis and treatment of pathogenic **neisseria** and other microorganisms having type 4 pilin
IN Normark, Staffan, Clayton, MO, United States
Jonsson, Ann-Beth, Umea, Sweden
PA Washington University, St. Louis, MO, United States (U.S. corporation)
PI US 5834591 19981110
AI US 1995-415788 19950403 (8)
RLI Continuation of Ser. No. US 1992-829465, filed on 31 Jan 1992, now abandoned which is a continuation-in-part of Ser. No. US 1991-648781, filed on 31 Jan 1991, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Sidberry, Hazel F.
CLMN Number of Claims: 44
ECL Exemplary Claim: 1
DRWN 18 Drawing Figure(s); 18 Drawing Page(s)
LN.CNT 3804
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 15 OF 27 USPATFULL
AB Recombinant methods for recovering wildtype or engineered negative stranded, non-segmented RNA virus genomes containing non-coding 3' and 5' regions (e.g. leader or trailer regions) surrounding one, several or all of the genes of the virus or one or more heterologous gene(s) in the form of ribonucleocapsids containing N, P and L proteins, which are capable of replicating and assembling with the remaining structural proteins to bud and form virions, or which are only capable of infecting one cell, or are transcribing particles, are disclosed. Novel vaccines, gene therapy vectors and antiviral compounds based on these viral particles are also disclosed.
AN 1998:91857 USPATFULL
TI Stranded RNA virus particles
IN Wertz, Gail W., Birmingham, AL, United States
Yu, Qingzhong, Birmingham, AL, United States
Ball, Laurence A., Birmingham, AL, United States
Barr, John N., Birmingham, AL, United States
Whelan, Sean P. J., Birmingham, AL, United States
PA UAB Research Foundation, Birmingham, AL, United States (U.S. corporation)
PI US 5789229 19980804
AI US 1995-514975 19950929 (8)
RLI Continuation-in-part of Ser. No. US 1995-475587, filed on 7 Jun 1995, now abandoned which is a continuation-in-part of Ser. No. US 1994-316438, filed on 30 Sep 1994, now patented, Pat. No. US 5716821
DT Utility
FS Granted
EXNAM Primary Examiner: Chambers, Jasemine C.; Assistant Examiner: Clark,

Deborah J. R.
LREP Adler, Benjamin Aaron
CLMN Number of Claims: 15
ECL Exemplary Claim: 1,6,11
DRWN 7 Drawing Figure(s); 7 Drawing Page(s)
LN.CNT 1922
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 16 OF 27 USPATFULL
AB The present invention is directed to methods for increasing secretion of an overexpressed gene product present in a host cell, by inducing expression of chaperone proteins within the host cell.
AN 1998:75395 USPATFULL
TI Methods for increasing secretion of overexpressed proteins
IN Wittrup, Karl Dane, Urbana, IL, United States
Robinson, Anne Skaja, Champaign, IL, United States
PA Research Corporation Technologies, Inc., Tucson, AZ, United States (U.S. corporation)
PI US 5773245 19980630
AI US 1995-441139 19950515 (8)
RLI Continuation of Ser. No. US 1993-89997, filed on 6 Jul 1993, now abandoned which is a continuation of Ser. No. US 1992-956699, filed on 2 Oct 1992, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Bugaisky, Gabriele E.
LREP Scully, Scott, Murphy & Presser
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 1812
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 17 OF 27 USPATFULL
AB The present invention defines an assay useful for screening libraries of synthetic or biological compounds for their ability to bind specific **DNA** test sequences. The assay is also useful for determining the sequence specificity and relative **DNA**-binding affinity of **DNA**-binding molecules for any particular **DNA** sequence. Also described herein are potential applications of the assay, including: 1) the detection of lead compounds or new drugs via the mass screening of libraries of synthetic or biological compounds (i.e., fermentation broths); 2) the design of sequence-specific **DNA** -binding drugs comprised of homo- or hetero-meric subunits of molecules for which the sequence specificity was determined using the assay; and 3) the use of molecules for which sequence specificity was determined using the assay as covalently attached moieties to aid in the binding of nucleic acid or other macromolecular polymers to nucleic acid sequences.
AN 1998:44877 USPATFULL
TI Sequence-directed **DNA**-binding molecules compositions and methods
IN Edwards, Cynthia A., Menlo Park, CA, United States
Fry, Kirk E., Palo Alto, CA, United States
Cantor, Charles R., Boston, MA, United States
Andrews, Beth M., Maynard, MA, United States
PA Genelabs Technologies, Inc., Redwood City, CA, United States (U.S. corporation)
PI US 5744131 19980428
AI US 1995-476876 19950607 (8)
RLI Division of Ser. No. US 1992-996783, filed on 23 Dec 1992 which is a continuation-in-part of Ser. No. US 1991-723618, filed on 27 Jun 1991, now abandoned
DT Utility

FS Granted
EXNAM Primary Examiner: Zitomer, Stephanie W.; Assistant Examiner: Atzel, Amy
LREP Fabian, Gary R., Stratford, Carol A., Dehlinger, Peter J.
CLMN Number of Claims: 3
ECL Exemplary Claim: 1
DRWN 48 Drawing Figure(s); 33 Drawing Page(s)
LN.CNT 5113
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 18 OF 27 USPATFULL
AB The present invention defines an assay useful for screening libraries of synthetic or biological compounds for their ability to bind specific **DNA** test sequences. The assay is also useful for determining the sequence specificity and relative **DNA**-binding affinity of **DNA**-binding molecules for any particular **DNA** sequence. Also described herein are potential applications of the assay, including: 1) the detection of lead compounds or new drugs via the mass screening of libraries of synthetic or biological compounds (i.e., fermentation broths); 2) the design of sequence-specific **DNA** -binding drugs comprised of homo- or hetero-meric subunits of molecules for which the sequence specificity was determined using the assay; and 3) the use of molecules for which sequence specificity was determined using the assay as covalently attached moieties to aid in the binding of nucleic acid or other macromolecular polymers to nucleic acid sequences.
AN 1998:39383 USPATFULL
TI Sequence-directed **DNA**-binding molecules compositions and methods
IN Edwards, Cynthia A., Menlo Park, CA, United States
Fry, Kirk E., Palo Alto, CA, United States
Cantor, Charles R., Boston, MA, United States
Andrews, Beth M., Maynard, MA, United States
PA Genelabs Technologies, Inc., Redwood City, CA, United States (U.S. corporation)
PI US 5738990 19980414
AI US 1995-475221 19950607 (8)
RLI Division of Ser. No. US 1992-996783, filed on 23 Dec 1992 which is a continuation-in-part of Ser. No. US 1991-723618, filed on 27 Jun 1991, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Guzo, David; Assistant Examiner: Brusca, John S.
LREP Fabian, Gary R., Stratford, Carol A., Dehlinger, Peter J.
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 48 Drawing Figure(s); 33 Drawing Page(s)
LN.CNT 5040
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 19 OF 27 USPATFULL
AB The present invention defines an assay useful for screening libraries of synthetic or biological compounds for their ability to bind specific **DNA** test sequences. The assay is also useful for determining the sequence specificity and relative **DNA**-binding affinity of **DNA**-binding molecules for any particular **DNA** sequence. Also described herein are potential applications of the assay, including: 1) the detection of lead compounds or new drugs via the mass screening of libraries of synthetic or biological compounds (i.e., fermentation broths); 2) the design of sequence-specific **DNA** -binding drugs comprised of homo- or hetero-meric subunits of molecules for which the sequence specificity was determined using the assay; and 3) the use of molecules for which sequence specificity was determined using the assay as covalently attached moieties to aid in the binding of nucleic acid or other macromolecular polymers to nucleic acid sequences.
AN 1998:14634 USPATFULL

TI Method of constructing sequence-specific **DNA**-binding molecules
IN Edwards, Cynthia A., Menlo Park, CA, United States
Fry, Kirk E., Palo Alto, CA, United States
Cantor, Charles R., Boston, MA, United States
Andrews, Beth M., Watertown, MA, United States
PA Genelabs Technologies, Inc., Redwood City, CA, United States (U.S. corporation)
PI US 5716780 19980210
AI US 1995-484499 19950607 (8)
RLI Division of Ser. No. US 1992-996783, filed on 23 Dec 1992 which is a continuation-in-part of Ser. No. US 1991-723618, filed on 27 Jun 1991, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Jones, W. Gary; Assistant Examiner: Atzel, Amy
LREP Fabian, Gary R., Stratford, Carol A., Dehlinger, Peter J.
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 48 Drawing Figure(s); 33 Drawing Page(s)
LN.CNT 4929
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 20 OF 27 SCISEARCH COPYRIGHT 2002 ISI (R)
AB Members of the bacterial families Haemophilus and **Neisseria**, important human pathogens that commonly colonize the nasopharynx, are naturally competent for **DNA** uptake from their environment. In each genus this process is discriminant in favor of its own and against foreign **DNA** through sequence specificity of **DNA** receptors, The Haemophilus **DNA** uptake apparatus binds a 29-bp oligonucleotide domain containing a highly conserved 9-bp core sequence, whereas the neisserial apparatus binds a 10-bp motif, Each motif (''uptake sequence'', US) is highly over-represented in the chromosome of the corresponding genus, particularly concentrated with core sequences in inverted pairs forming gene terminators. Two Haemophilus core USs were unexpectedly found forming the terminator of sodC in **Neisseria meningitidis** (meningococcus), and sequence analysis strongly suggests that this virulence gene, located next to IS1106, arose through horizontal transfer from Haemophilus. By using USs as search strings in a computer-based analysis of genome sequence, it was established that while USs of the ''wrong'' genus do not occur commonly in **Neisseria** or Haemophilus, where they do they are highly likely to flag domains of chromosomal **DNA** that have been transferred from Haemophilus. Three independent domains of Haemophilus-like **DNA** were found in the meningococcal chromosome, associated respectively with the virulence gene sodC, the bio gene cluster, and an unidentified orf. This report identifies inter-generically transferred **DNA** and its source in bacteria, and further identifies transformation with heterologous chromosomal **DNA** as a way of establishing potentially important chromosomal mosaicism in these pathogenic bacteria.
AN 1998:810288 SCISEARCH
GA The Genuine Article (R) Number: 129DL
TI Natural genetic exchange between Haemophilus and **Neisseria**: Intergeneric transfer of chromosomal genes between major human pathogens
AU Kroll J S (Reprint); Wilks K E; Farrant J L; Langford P R
CS UNIV LONDON IMPERIAL COLL SCI TECHNOL & MED, ST MARYS HOSP, MOL INFECT DIS GRP, DEPT PAEDIAT, LONDON W2 1PG, ENGLAND (Reprint)
CYA ENGLAND
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (13 OCT 1998) Vol. 95, No. 21, pp. 12381-12385.
Publisher: NATL ACAD SCIENCES, 2101 CONSTITUTION AVE NW, WASHINGTON, DC 20418.
ISSN: 0027-8424.
DT Article; Journal
FS LIFE

LA English
REC Reference Count: 41
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L10 ANSWER 21 OF 27 MEDLINE
AB Meningococcal sodC encodes periplasmic copper- and zinc-cofactored **superoxide dismutase** (Cu,Zn SOD) which catalyzes the conversion of the superoxide radical anion to hydrogen peroxide, preventing a sequence of reactions leading to production of toxic hydroxyl free radicals. From its periplasmic location, Cu,Zn SOD was inferred to acquire its substrate from outside the bacterial cell and was speculated to play a role in preserving meningococci from the action of microbicidal oxygen free radicals produced in the context of host defense. A sodC mutant was constructed by allelic exchange and was used to investigate the role of Cu,Zn SOD in pathogenicity. Wild-type and mutant meningococci grew at comparable rates and survived equally long in aerobic liquid culture. The mutant showed no increased sensitivity to paraquat, which generates superoxide within the cytosol, but was approximately 1,000-fold more sensitive to the toxicity of superoxide generated in solution by the xanthine/xanthine oxidase system. These data support a role for meningococcal Cu,Zn SOD in protection against exogenous superoxide. In experiments to translate this into a role in pathogenicity, wild-type and mutant organisms were used in an intraperitoneal mouse infection model. The sodC mutant was significantly less virulent. We conclude that periplasmic Cu,Zn SOD contributes to the virulence of **Neisseria meningitidis**, most likely by reducing the effectiveness of toxic oxygen host defenses.
AN 1998084476 MEDLINE
DN 98084476 PubMed ID: 9423860
TI Periplasmic **superoxide dismutase** in meningococcal pathogenicity.
AU Wilks K E; Dunn K L; Farrant J L; Reddin K M; Gorringe A R; Langford P R; Kroll J S
CS Department of Paediatrics, Imperial College, School of Medicine at St. Mary's Hospital, London, United Kingdom.
SO INFECTION AND IMMUNITY, (1998 Jan) 66 (1) 213-7.
Journal code: 0246127. ISSN: 0019-9567.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AJ001313
EM 199801
ED Entered STN: 19980206
Last Updated on STN: 20000303
Entered Medline: 19980127

L10 ANSWER 22 OF 27 USPATFULL
AB In order to obtain a novel binding protein against a chosen target, **DNA** molecules, each encoding a protein comprising one of a family of similar potential binding domains and a structural signal calling for the display of the protein on the outer surface of a chosen bacterial cell, bacterial spore or phage (genetic package) are introduced into a genetic package. The protein is expressed and the potential binding domain is displayed on the outer surface of the package. The cells or viruses bearing the binding domains which recognize the target molecule are isolated and amplified. The successful binding domains are then characterized. One or more of these successful binding domains is used as a model for the design of a new family of potential binding domains, and the process is repeated until a novel binding domain having a desired affinity for the target molecule is obtained. In one embodiment, the first family of potential binding domains is related to bovine pancreatic trypsin inhibitor, the genetic package is M13 phage, and the protein includes the outer surface

transport signal of the M13 gene III protein.
AN 96:101466 USPATFULL
TI Directed evolution of novel binding proteins
IN Ladner, Robert C., Ijamsville, MD, United States
Guterman, Sonia K., Belmont, MA, United States
Roberts, Bruce L., Milford, MA, United States
Markland, William, Milford, MA, United States
Ley, Arthur C., Newton, MA, United States
Kent, Rachel B., Boxborough, MA, United States
PA Protein Engineering Corporation, Cambridge, MA, United States (U.S. corporation)
PI US 5571698 19961105
AI US 1993-57667 19930618 (8)
DCD 20100629
RLI Continuation of Ser. No. US 1991-664989, filed on 1 Mar 1991, now patented, Pat. No. US 5223409 which is a continuation-in-part of Ser. No. US 1990-487063, filed on 2 Mar 1990, now abandoned which is a continuation-in-part of Ser. No. US 1988-240160, filed on 2 Sep 1988, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Ulm, John
LREP Cooper, Iver P.
CLMN Number of Claims: 83
ECL Exemplary Claim: 1
DRWN 16 Drawing Figure(s); 16 Drawing Page(s)
LN.CNT 15323
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 23 OF 27 SCISEARCH COPYRIGHT 2002 ISI (R)
AB Antioxidant enzymes are thought to be important for the survival of pathogenic **Neisseria** species. We have further characterized the glutathione peroxidase homolog gene (gpxA), which we recently isolated from **Neisseria meningitidis** FAM20 (T. D. E, Moore and P. F. Sparling, Infect. Immun. 63:1603-1607, 1995). GpxA was found to be produced constitutively in vivo. An isogenic, omega insertion mutant in the gpxA. gene was constructed and characterized. The gpxA insertion mutant was much more sensitive to the oxidative stress caused by paraquat and slightly more sensitive to hydrogen peroxide. This is the first demonstration of a phenotype arising from a mutation of a glutathione peroxidase homolog gene in a prokaryotic organism. Protection of the cell by GpxA from the effects of oxidative stress caused by aerobic metabolism may contribute to the ability of **Neisseria meningitidis** to cause disease in the human host.
AN 96:534599 SCISEARCH
GA The Genuine Article (R) Number: UW795
TI INTERRUPTION OF THE GPXA GENE INCREASES THE SENSITIVITY OF **NEISSERIA-MENINGITIDIS** TO PARAQUAT
AU MOORE T D E (Reprint); SPARLING P F
CS UNIV N CAROLINA, LINEBERGER COMPREHENS CANC CTR, CHAPEL HILL, NC, 27599 (Reprint); UNIV N CAROLINA, DEPT MED, CHAPEL HILL, NC, 27599; UNIV N CAROLINA, DEPT MICROBIOL & IMMUNOL, CHAPEL HILL, NC, 27599
CYA USA
SO JOURNAL OF BACTERIOLOGY, (JUL 1996) Vol. 178, No. 14, pp. 4301-4305. ISSN: 0021-9193.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 42
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L10 ANSWER 24 OF 27 USPATFULL
AB In order to obtain a novel binding protein against a chosen target, **DNA** molecules, each encoding a protein comprising one of a

family of similar potential binding domains and a structural signal calling for the display of the protein on the outer surface of a chosen bacterial cell, bacterial spore or phage (genetic package) are introduced into a genetic package. The protein is expressed and the potential binding domain is displayed on the outer surface of the package. The cells or viruses bearing the binding domains which recognize the target molecule are isolated and amplified. The successful binding domains are then characterized. One or more of these successful binding domains is used as a model for the design of a new family of potential binding domains, and the process is repeated until a novel binding domain having a desired affinity for the target molecule is obtained. In one embodiment, the first family of potential binding domains is related to bovine pancreatic trypsin inhibitor, the genetic package is M13 phage, and the protein includes the outer surface transport signal of the M13 gene III protein.

AN 95:29292 USPATFULL
TI Viruses expressing chimeric binding proteins
IN Ladner, Robert C., Ijamsville, MD, United States
Guterman, Sonia K., Belmont, MA, United States
Roberts, Bruce L., Milford, MA, United States
Markland, William, Milford, MA, United States
Ley, Arthur C., Newton, MA, United States
Kent, Rachel B., Boxborough, MA, United States
PA Protein Engineering Corporation, Cambridge, MA, United States (U.S. corporation)
PI US 5403484 19950404
AI US 1993-9319 19930126 (8)
RLI Division of Ser. No. US 1991-664989, filed on 1 Mar 1991, now patented, Pat. No. US 5223409 which is a continuation-in-part of Ser. No. US 1990-487063, filed on 2 Mar 1990, now abandoned which is a continuation-in-part of Ser. No. US 1988-240160, filed on 2 Sep 1988, now abandoned
PRAI WO 1989-3731 19890901
DT Utility
FS Granted
EXNAM Primary Examiner: Hill, Jr., Robert J.; Assistant Examiner: Ulm, John D.
LREP Cooper, Iver P.
CLMN Number of Claims: 49
ECL Exemplary Claim: 1
DRWN 16 Drawing Figure(s); 16 Drawing Page(s)
LN.CNT 14368
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 25 OF 27 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1995:290444 BIOSIS
DN PREV199598304744
TI The iron-cofactored **superoxide dismutase** from pathogenic **Neisseria**: Cloning and characterization of the sodB gene.
AU Karkhoff-Schweizer, R. R. (1); Schweizer, H. P.; Hassett, D. J.
CS (1) Univ. Calgary Health Sci. Cent., Calgary, AB Canada
SO Abstracts of the General Meeting of the American Society for Microbiology, (1995) Vol. 95, No. 0, pp. 196.
Meeting Info.: 95th General Meeting of the American Society for Microbiology Washington, D.C., USA May 21-25, 1995
ISSN: 1060-2011.
DT Conference
LA English

L10 ANSWER 26 OF 27 USPATFULL
AB In order to obtain a novel binding protein against a chosen target, **DNA** molecules, each encoding a protein comprising one of a family of similar potential binding domains and a structural signal calling for the display of the protein on the outer surface of a chosen

bacterial cell, bacterial spore or phage (genetic package) are introduced into a genetic package. The protein is expressed and the potential binding domain is displayed on the outer surface of the package. The cells or viruses bearing the binding domains which recognize the target molecule are isolated and amplified. The successful binding domains are then characterized. One or more of these successful binding domains is used as a model for the design of a new family of potential binding domains, and the process is repeated until a novel binding domain having a desired affinity for the target molecule is obtained. In one embodiment, the first family of potential binding domains is related to bovine pancreatic trypsin inhibitor, the genetic package is M13 phage, and the protein includes the outer surface transport signal of the M13 gene III protein.

AN 93:52487 USPATFULL
TI Directed evolution of novel binding proteins
IN Ladner, Robert C., Ijamsville, MD, United States
Guterman, Sonia K., Belmont, MA, United States
Roberts, Bruce L., Milford, MA, United States
Markland, William, Milford, MA, United States
Ley, Arthur C., Newton, MA, United States
Kent, Rachel B., Boxborough, MA, United States
PA Protein Engineering Corp., Cambridge, MA, United States (U.S. corporation)
PI US 5223409 19930629
AI US 1991-664989 19910301 (7)
RLI Continuation-in-part of Ser. No. US 1990-487063, filed on 2 Mar 1990, now abandoned And a continuation-in-part of Ser. No. US 1988-240160, filed on 2 Sep 1988, now abandoned
DT Utility
FS Granted
EXNAM Primary Examiner: Hill, Jr., Robert J.; Assistant Examiner: Ulm, John D.
LREP Cooper, Iver P.
CLMN Number of Claims: 66
ECL Exemplary Claim: 1
DRWN 16 Drawing Figure(s); 16 Drawing Page(s)
LN.CNT 15410
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L10 ANSWER 27 OF 27 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AB Manganese **superoxide dismutase** (the sodA gene product) in Escherichia coli, is negatively regulated by two global regulators, ArcA (aerobic respiration control) and Fur (ferric uptake regulation), coupling its expression to aerobic metabolic and the intracellular iron pool. Footprinting analyses were carried out on the sodA promoter region with purified Fur protein and with ArcA protein overproduced in crude extracts. ArcA is ale to bind in vitro in the absence of the in vivo triggering signal. The binding occurs in one step and study of contact within the operator sequence reveals binding on one side of the double helix. The **DNA** protection extends to a much larger region (about 65 bp) than would be expected for a 27 kDa protein, suggesting polymerization. Both Fur and ArcA footprints encompass the -35 and -10 promoter region and there is considerable overlap on their binding sequences, in agreement with in vivo results suggesting that either regulator alone can block sodA transcription. Furthermore, competition experiments show that Fur and ArcA binding to the sodA promoter are mutually exclusive and that ArcA can easily displace Fur, but not vice versa. The biological significance of this in vitro behaviour is discussed.
AN 1993:432264 BIOSIS
DN PREV199396086889
TI Iron and oxygen regulation of Escherichia coli magnesium **superoxide dismutase** expression: Competition between the global regulators Fur and ArcA for binding to **DNA**.
AU Tardat, Brigitte; Touati, Daniele (1)

CS (1) Inst. Jacques Monod, CNRS, Univ. Paris 7, 2 Place Jussieu, 75251 Paris Cedex 05 France
SO Molecular Microbiology, (1993) Vol. 9, No. 1, pp. 53-63.
ISSN: 0950-382X.
DT Article
LA English

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L14 ANSWER 34 OF 158 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 1

AB **Haemophilus** ducreyi produces a periplasmic copper-zinc **superoxide dismutase** (Cu-Zn SOD), which is thought to protect the organism from exogenous reactive oxygen species generated by neutrophils during an inflammatory response. We had previously identified the gene, sodC, responsible for the production and secretion of Cu-Zn SOD and constructed an isogenic H. ducreyi strain with a mutation in the sodC gene (35000HP-sodC-cat). Compared to the parent, the mutant does not survive in the presence of exogenous superoxide (L. R. San Mateo, M. Hobbs, and T. H. Kawula, Mol. Microbiol. 27:391-404, 1998) and is impaired in the swine model of H. ducreyi infection (L. R. San Mateo, K. L. Toffer, P. E. Orndorff, and T. H. Kawula, Infect. Immun. 67:5345-5351, 1999). To test whether Cu-Zn SOD is important for bacterial survival in vivo, six human volunteers were experimentally infected with 35000HP and 35000HP-sodC-cat and observed for papule and pustule formation. Papules developed at similar rates at sites inoculated with the mutant or parent. The pustule formation rates were 75% (95% confidence intervals (CI), 43 to 95%) at 12 parent-inoculated sites and 67% (95% CI, 41 to 88%) at 18 mutant-inoculated sites (P = 0.47). There was no significant difference in levels of H. ducreyi recovery from mutant- and parent-inoculated biopsy sites. These results suggest that expression of Cu-Zn SOD does not play a major role in the survival of this pathogen in the initial stages of experimental infection of humans.

AN 2002:197250 BIOSIS
DN PREV200200197250

TI A **superoxide dismutase** C mutant of **Haemophilus** ducreyi is virulent in human volunteers.

AU Bong, Cliffton T. H.; Fortney, Kate R.; Katz, Barry P.; Hood, Antoinette F.; San Mateo, Lani R.; Kawula, Thomas H.; Spinola, Stanley M. (1)
CS (1) Indiana University, 545 Barnhill Dr., 435 Emerson Hall, Indianapolis, IN, 46202-5124: sspinola@iupui.edu USA

SO Infection and Immunity, (March, 2002) Vol. 70, No. 3, pp. 1367-1371. print.
ISSN: 0019-9567.
DT Article
LA English

L14 ANSWER 35 OF 158 SCISEARCH COPYRIGHT 2002 ISI (R)

AB Periplasmic copper- and zinc-cofactored **superoxide dismutases** ([Cu,Zn]-SODs, SodC) of several Gram-negative pathogens can protect against superoxide-radical-mediated host defences, and thus contribute to virulence. This role has been previously defined for one [Cu,Zn]-SOD in various Salmonella serovars. Following the recent discovery of a second periplasmic [Cu,Zn]-SOD in Salmonella, the effect of knockout mutations in one or both of the original sodC-1 and the new sodC-2 on the virulence of the porcine pathogen Salmonella choleraesuis is investigated here. In comparison to wild-type, while sodC mutants - whether single or double - showed no impairment in growth, they all showed equally enhanced sensitivity to superoxide and a dramatically increased sensitivity to the combination of superoxide and nitric oxide in vitro. This observation had its correlate in experimental infection both ex vivo and in vivo, Mutation of sodC significantly impaired survival of S. choleraesuis in interferon gamma-stimulated murine macrophages compared to wild-type organisms, and all S. choleraesuis sodC mutants persisted in significantly lower numbers than wild-type in BALB/c (Ity(s)) and C3H/HeN (Ity(r)) mice after experimental infection, but in no experimental system were sodC-1 sodC-2 double mutants more attenuated than either single mutant. These data suggest that both [Cu,Zn]-SODs are needed to protect bacterial periplasmic or membrane components. While SodC plays a role in S. choleraesuis virulence, the data presented here suggest that this is through overcoming a threshold effect, probably achieved by acquisition of sodC-1 on a bacteriophage. Loss of either sodC gene confers maximum vulnerability to superoxide on S. choleraesuis.

AN 2002:262769 SCISEARCH
GA The Genuine Article (R) Number: 529YB

TI The role of two periplasmic copper- and zinc-cofactored **superoxide dismutases** in the virulence of Salmonella choleraesuis

AU Sansone A; Watson P R; Wallis T S; Langford P R; Kroll J S (Reprint)
CS Univ London Imperial Coll Sci Technol & Med, Fac Med, Dept Paediat, Mol Infect Dis Grp, St Marys Hosp Campus, Norfolk Pl, London W2 1PG, England (Reprint); Univ London Imperial Coll Sci Technol & Med, Fac Med, Dept Paediat, Mol Infect Dis Grp, London W2 1PG, England; Inst Anim Hlth, Newbury RG20 7NN, Berks, England
CYA England

SO MICROBIOLOGY-SGM, (MAR 2002) Vol. 148, Part 3, pp. 719-726.
Publisher: SOC GENERAL MICROBIOLOGY, MARLBOROUGH HOUSE, BASINGSTOKE RD, SPENCERS WOODS, READING RG7 1AE, BERKS, ENGLAND.
ISSN: 1350-0872.
DT Article; Journal
LA English
REC Reference Count: 37

ANSWER 36 OF 158 CABA COPYRIGHT 2002 CABI DUPLICATE 2

AB Several cryptic genospecies, capsulate division I and II strains (including biotype IV strains), and a defined collection of noncapsulate **Haemophilus** influenzae sensu stricto strains were screened for the presence of sodC and the ability to produce copper (Cu) zinc (Zn) **superoxide dismutase** (SOD) activity. Results showed that the presence of active Cu- and Zn-containing SOD in isolates of the cryptic genospecies of **Haemophilus**, responsible for urogenital, neonatal, and mother-infant infections, can be used as a biochemical marker to discriminate them from H. influenzae sensu stricto strains.

AN 2002:54815 CABA

DN 20023017963

TI Active copper- and zinc-containing **superoxide dismutase** in the cryptic genospecies of **Haemophilus** causing urogenital and neonatal infections discriminates them from **Haemophilus** influenzae sensu stricto

AU Langford, P. R.; Sheehan, B. J.; Taheed Shaikh; Kroll, J. S.

CS Molecular Infectious Diseases Group, Department of Paediatrics, Faculty of Medicine, Imperial College, St. Mary's Campus, London W2 1PG, UK.

SO Journal of Clinical Microbiology, (2002) Vol. 40, No. 1, pp. 268-270. 21 ref.
ISSN: 0095-1137

DT Journal

LA English

4 ANSWER 50 OF 158 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 3

AB **Haemophilus** ducreyi, the causative agent of the genital ulcerative disease known as chancroid, is unable to synthesize heme, which it acquires from humans, its only known host. Here we provide evidence that the periplasmic Cu,Zn-**superoxide dismutase** from this organism is a heme-binding protein, unlike all the other known Cu,Zn-**superoxide dismutases** from bacterial and eukaryotic species. When the H. ducreyi enzyme was expressed in Escherichia coli cells grown in standard LB medium, it contained only limited amounts of heme covalently bound to the polypeptide but was able efficiently to bind exogenously added hemin. Resonance Raman and electronic spectra at neutral pH indicate that H. ducreyi Cu,Zn-**superoxide dismutase** contains a 6-coordinated low spin heme, with two histidines as the most likely axial ligands. By site-directed mutagenesis and analysis of a structural model of the enzyme, we identified as a putative axial ligand a histidine residue (His-64) that is present only in the H. ducreyi enzyme and that was located at the bottom of the dimer interface. The introduction of a histidine residue in the corresponding position of the Cu,Zn-**superoxide dismutase** from **Haemophilus** parainfluenzae was not sufficient to confer the ability to bind heme, indicating that other residues neighboring His-64 are involved in the formation of the heme-binding pocket. Our results suggest that periplasmic Cu,Zn-**superoxide dismutase** plays a role in heme metabolism of H. ducreyi and provide further evidence for the structural flexibility of bacterial enzymes of this class.

AN 2001:440169 BIOSIS

DN PREV200100440169

TI A novel heme protein, the Cu,Zn-**superoxide dismutase** from **Haemophilus** ducreyi.

AU Pacello, Francesca; Langford, Paul R.; Kroll, J. Simon; Indiani, Chiara; Smulevich, Giulietta; Desideri, Alessandro; Rotilio, Giuseppe; Battistoni, Andrea (1)

CS (1) Dip. di Biologia, Universita di Roma "Tor Vergata", Via della Ricerca Scientifica, 00133, Roma: andrea.battistoni@uniroma2.it Italy

SO Journal of Biological Chemistry, (August 10, 2001) Vol. 276, No. 32, pp. 30326-30334. print.
ISSN: 0021-9258.

DT Article

LA English

SL English

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L14 ANSWER 137 OF 158 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 17

AB Oxygen free radicals present a serious potential threat to microbial survival, through their ability to inflict indiscriminate damage on proteins and DNA. **Superoxide dismutase** (SOD, EC 1.15.1.1), among other oxygen-metabolizing enzymes, is essential to prevent these toxic molecules from accumulating in the bacterial cytosol during aerobic metabolism. The gene sodA, encoding manganese-containing SOD ((Mn)-SOD), has been cloned from a virulent strain of **Haemophilus** influenzae type b using degenerate oligonucleotides encoding regions of the gene conserved across different bacterial species. The gene product has been identified as (Mn)-SOD by its similarity at key amino acid residues to known examples of the enzyme, by expression of enzymatically active protein from cloned DNA expressed in Escherichia coli, and by demonstration that an in-frame deletion in the gene abolishes this activity. In contrast to the situation in E. coli, this (Mn)-SOD is the only active SOD detected in H. influenzae. In further contrast to E. coli, (Mn)-SOD gene expression in H. influenzae has been found to be only partially repressed under anaerobic conditions. When expressed in E. coli the gene is regulated by Fur and Fnr, and the promoter region, identified experimentally, has been found to contain nucleotide sequence motifs similar to the Fur- and Fnr-binding sequences of E. coli, suggesting the involvement of analogues of these aerobiosis-responsive activators in H. influenza gene expression.

AN 1994:60656 BIOSIS
DN PREV199497073656

TI Molecular and genetic characterization of **superoxide dismutase** in **Haemophilus** influenzae type b.

AU Kroll, J. S. (1); Langford, P. R.; Saah, J. R.; Loynds, B. M.
CS (1) Mol. Infectious Diseases Group, Dep. Paediatrics, Inst. Mol. Med., John Radcliffe Hosp., Oxford OX3 9DU UK

SO Molecular Microbiology, (1993) Vol. 10, No. 4, pp. 839-848.
ISSN: 0950-382X.
DT Article
LA English

ANSWER 129 OF 158 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 16

AB Copper- and zinc-containing **superoxide dismutases** ((Cu,Zn)-SODs) are generally considered almost exclusively eukaryotic enzymes, protecting the cytosol and extracellular compartments of higher organisms from damage by oxygen free-radicals. The recent description of a few examples of bacterial forms of the enzyme, located in the periplasm of different Gram-negative micro-organisms, prompted a re-evaluation of this general perception. A PCR-based approach has been developed and used successfully to identify bacterial genes encoding (Cu,Zn)-SOD in a wide range of important human and animal pathogens - members of the **Haemophilus**, Actinobacillus and Pasteurella (HAP) group, and Neisseria meningitidis. Comparison of (Cu,Zn)-SOD peptide sequences found in **Haemophilus** ducreyi, Actinobacillus pleuropneumoniae, Actinobacillus actinomycetemcomitans, Pasteurella multocida, and N. meningitidis with previously described bacterial proteins and examples of eukaryotic (Cu,Zn)-SOD has shown that the bacterial proteins constitute a distinct family apparently widely separated in evolutionary terms from the eukaryotic examples. The widespread occurrence of (Cu,Zn)-SOD in the periplasm of bacterial pathogens, appropriately located to dismute exogenously derived superoxide radical anions, suggests that this enzyme may play a role in the interactive biology of organisms with their hosts and so contribute to their capacity to cause disease.

AN 1995:532743 BIOSIS
DN PREV199598547043

TI Bacterial (Cu,Zn)-**superoxide dismutase**: Phylogenetically distinct from the eukaryotic enzyme, and not so rare after all.

AU Kroll, J. Simon (1); Langford, Paul R.; Wilks, Kathryn E.; Keil, Anthony D.
CS (1) Mol. Infect. Dis. Group, Dep. Paediatr., Imperial Coll. Sci. Technol. Med., St. Mary's Hosp., London W2 1PG UK

SO Microbiology (Reading), (1995) Vol. 141, No. 9, pp. 2271-2279. ISSN: 1350-0872.
DT Article

14 ANSWER 127 OF 158 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 14

AB The sodC gene of **Haemophilus** ducreyi was cloned and sequenced. The deduced amino acid sequence of this protein exhibited 71.6% identity and 81.8% similarity to the H. influenzae and H. parainfluenzae copper (Cu), zinc (Zn)-**superoxide dismutase** (SOD) enzymes. This gene was localized to a 2.2-kb H. ducreyi chromosomal DNA insert in plasmid pHdSOD. SOD activity was expressed in cell-free extracts of Escherichia coli containing the recombinant plasmid pHdSOD and was localized to the periplasmic space. The Cu,Zn-SOD produced by the H. ducreyi sodC gene did not complement the aerobic growth defect of an E. coli SOD-deficient mutant.

AN 1997:64969 BIOSIS
DN PREV199799364172

TI Cloning and sequencing of the gene encoding the Cu,Zn-**superoxide dismutase** of **Haemophilus** ducreyi.

AU Stevens, Marla K.; Hassett, Daniel J.; Radolf, Justin D.; Hansen, Eric J. (1)
CS (1) Dep. Microbiol., Univ. Texas Southwestern Med. Cent., 6000 Harry Hines Blvd., Dallas, TX 75235-9048 USA

SO Gene (Amsterdam), (1996) Vol. 183, No. 1-2, pp. 35-40.
ISSN: 0378-1119.
DT Article
LA English

4 ANSWER 124 OF 158 SCISEARCH COPYRIGHT 2002 ISI (R)

AB Copper-zinc **superoxide dismutases** (Cu,Zn SODs), until recently considered very unusual in bacteria, are now being found in a wide range of gram-negative bacterial species, Here we report the cloning and characterization of sodC, encoding Cu,Zn SOD in Actinobacillus pleuropneumoniae, a major pathogen of pigs and the causative organism of porcine pleuropneumonia. sodC was shown to lie on a monocistronic operon, at the chromosomal locus between the genes asd (encoding aspartate semialdehyde dehydrogenase) and recF. The primary gene product was shown to have an N-terminal peptide extension functioning as a leader peptide, so that the mature Actinobacillus enzyme, like other bacterial examples, is directed to the periplasm, where it is appropriately located to dismutate exogenously generated superoxide, While the role of these secreted bacterial SODs is unknown, we speculate that in A. pleuropneumoniae the enzyme may confer survival advantage by accelerating dismutation of superoxide derived from neutrophils, a central host defense response in the course of porcine infection.

AN 96:879178 SCISEARCH
GA The Genuine Article (R) Number: VU635
TI Cloning and molecular characterization of Cu,Zn **superoxide dismutase** from Actinobacillus pleuropneumoniae
AU Langford P R; Loynds B M; Kroll J S (Reprint)
CS ST MARYS HOSP, IMPERIAL COLL, SCH MED, MOL INFECT DIS GRP, LONDON W2 1PG, ENGLAND (Reprint); ST MARYS HOSP, IMPERIAL COLL, SCH MED, MOL INFECT DIS GRP, LONDON W2 1PG, ENGLAND
CYA ENGLAND
SO INFECTION AND IMMUNITY, (DEC 1996) Vol. 64, No. 12, pp. 5035-5041.
Publisher: AMER SOC MICROBIOLOGY, 1325 MASSACHUSETTS AVENUE, NW, WASHINGTON, DC 20005-4171.
ISSN: 0019-9567.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 57

4 ANSWER 105 OF 158 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 7

AB **Haemophilus** ducreyi causes chancroid, a sexually transmitted genital ulcer disease implicated in increased heterosexual transmission of HIV. As part of an effort to identify H. ducreyi gene products involved in virulence and pathogenesis, we created random TnphoA insertion mutations in an H. ducreyi 35 000 library cloned in Escherichia coli. Inserts encoding exported or secreted PhoA fusion proteins were characterized by DNA sequencing. One such clone encoded a Cu-Zn **superoxide dismutase** (SOD) enzyme. The Cu-Zn SOD was periplasmic in H. ducreyi and accounted for most of the detectable SOD activity in whole-cell lysates of H. ducreyi grown in vitro. To investigate the function of the Cu-Zn SOD, we created a Cu-Zn SOD-deficient H. ducreyi strain by inserting a cat cassette into the sodC gene. The wild-type and Cu-Zn SOD null mutant strains were equally resistant to excess cytoplasmic superoxide induced by paraquat, demonstrating that the Cu-Zn SOD did not function in the detoxification of cytoplasmic superoxide. However, the Cu-Zn SOD null strain was significantly more susceptible to killing by extracellular superoxide than the wild type. This result suggests that the H. ducreyi Cu-Zn SOD may play a role in bacterial defence against oxidative killing by host immune cells during infection.

AN 1998:122627 BIOSIS

DN PREV199800122627

TI Periplasmic copper-zinc **superoxide dismutase** protects **Haemophilus** ducreyi from exogenous superoxide.

AU San Mateo, Lani R.; Hobbs, Marcia M.; Kawula, Thomas H. (1)

CS (1) Dep. Microbiol. Immunol., Univ. North Carolina Sch. Med., Chapel Hill, NC 27599 USA

SO Molecular Microbiology, (Jan., 1998) Vol. 27, No. 2, pp. 391-404. ISSN: 0950-382X.

DT Article

LA English

5 ANSWER 1 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

AB Haemophilus influenzae type b (Hib) capsular polysaccharide (polyribosylribitol phosphate, PRP) is the active component of conjugate vaccines that have proven successful in preventing invasive Hib disease. Conjugation of PRP to a protein carrier greatly improves its immunogenicity providing protection in infants and subsequent antibody maturation upon boosting. In this study, fimbriae isolated from Bordetella pertussis have been assessed as novel carrier proteins. These proteins are components of some acellular pertussis vaccines and clinical trials have indicated that fimbriae could be important protective antigens against whooping cough. Fimbriae (Fim2 and Fim3) purified from B. pertussis were dissociated in 6 M guanidine hydrochloride, pH 10.5, to produce proteins of defined size and to facilitate the production and characterisation of the conjugates. Both carbodiimide-mediated coupling and reductive amination were used to conjugate PRP to dissociated fimbriae. Efficiency of conjugation was determined by size exclusion chromatography followed by protein and polysaccharide analysis of fractionated components. Immunisation of rabbits with dissociated fimbriae-PRP conjugates (D.fim-PRP) produced high anti-fimbrial and anti-PRP IgG titres. Use of a D.fim-PRP conjugate could protect against Hib disease and may also augment protection against B. pertussis.

AN 2001:309263 BIOSIS
DN PREV200100309263
TI Formulation and characterisation of Bordetella pertussis fimbriae as novel carrier proteins for Hib conjugate vaccines.
AU Crowley-Luke, Annette (1); Reddin, Karen; **Gorringe, Andrew**; Hudson, Michael J.; Robinson, Andrew
CS (1) Centre for Applied Microbiology and Research, Salisbury, SP4 0JG: annette.crowley-luke@camr.org.uk UK
SO Vaccine, (14 May, 2001) Vol. 19, No. 25-26, pp. 3399-3407. print. ISSN: 0264-410X.
DT Article
LA English
SL English

L15 ANSWER 2 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE 1

AB To better characterize the vaccine potential of Neisseria meningitidis transferrin binding proteins (Tbps), we have overexpressed TbpA and TbpB from Neisseria meningitidis isolate K454 in Escherichia coli. The ability to bind human transferrin was retained by both recombinant proteins, enabling purification by affinity chromatography. The recombinant Tbps were evaluated individually and in combination in a mouse intraperitoneal-infection model to determine their ability to protect against meningococcal infection and to induce cross-reactive and bactericidal antibodies. For the first time, TbpA was found to afford protection against meningococcal challenge when administered as the sole immunogen. In contrast to the protection conferred by TbpB, this protection extended to a serogroup C isolate and strain B16B6, a serogroup B isolate with a lower-molecular-weight TbpB than that from strain K454. However, serum from a TbpB-immunized rabbit was found to be significantly more bactericidal than that from a TbpA-immunized animal. Our evidence demonstrates that TbpA used as a vaccine antigen may provide protection against a wider range of meningococcal strains than does TbpB alone. This protection appears not to be due to complement-mediated lysis and indicates that serum bactericidal activity may not always be the most appropriate predictor of efficacy for protein-based meningococcal vaccines.

AN 2001:194558 BIOSIS
DN PREV200100194558
TI Recombinant Neisseria meningitidis transferrin binding protein A protects against experimental meningococcal infection.
AU West, David; Reddin, Karen; Matheson, Mary; Heath, Robert; Funnell, Simon; Hudson, Michael; Robinson, Andrew; **Gorringe, Andrew (1)**
CS (1) CAMR, Salisbury, SP4 0JG: andrew.gorringe@camr.org.uk UK

Publisher: Marcel Dekker, Inc. 270 Madison Avenue, New York, New York 10016, USA.
ISSN: 1043-2981. ISBN: 0-8247-9186-X.
DT Book
LA English

L20 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2002 ACS
AB A review, with 121 refs.
AN 1994:652598 CAPLUS
DN 121:252598
TI Molecular aspects of the pathogenesis of invasive Haemophilus influenzae type b infections
AU **Kroll, John Simon**; Langford, Paul Richard
CS St. Mary's Hospital Medical School, London, UK
SO Infectious Disease and Therapy (1994), 11(DEVELOPMENT AND CLINICAL USES OF HAEMOPHILUS B CONJUGATE VACCINES), 145-77
CODEN: IDTHER; ISSN: 1043-2981
DT Journal; General Review
LA English

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(FILE 'HOME' ENTERED AT 11:27:24 ON 17 OCT 2002)

     FILE 'BIOSIS, CABA, CAPLUS, EMBASE, LIFESCI, MEDLINE, SCISEARCH,
     USPATFULL, JAPIO' ENTERED AT 11:29:16 ON 17 OCT 2002
L1       141614 S (SUPEROXIDE DISMUTASE)
L2        69199 S NEISSERIA
L3        28553 S MENINGITIDIS
L4        28027 S L2 AND L3
L5           64 S L1 AND L4
L6           20 S L5 AND (IMMUNIZE OR VACCINAT? OR INJECT?)
L7           20 DUP REM L6 (0 DUPLICATES REMOVED)

     FILE 'STNGUIDE' ENTERED AT 11:34:40 ON 17 OCT 2002

     FILE 'BIOSIS, CABA, CAPLUS, EMBASE, LIFESCI, MEDLINE, SCISEARCH,
     USPATFULL, JAPIO' ENTERED AT 11:35:51 ON 17 OCT 2002
L8        13851 S L1 AND (NUCLEIC ACID MOLECULE OR DNA)
L9           27 S L8 AND L4
L10          27 DUP REM L9 (0 DUPLICATES REMOVED)

     FILE 'BIOSIS, CABA, CAPLUS, EMBASE, LIFESCI, MEDLINE, SCISEARCH,
     USPATFULL, JAPIO' ENTERED AT 11:41:54 ON 17 OCT 2002
L11         840 S L1 AND VACCINE
L12          27 S L11 AND L3
L13          27 DUP REM L12 (0 DUPLICATES REMOVED)
L14           5 S GORRINGE, ANDREW/AU
L15           4 DUP REM L14 (1 DUPLICATE REMOVED)
L16         102 S ROBINSON, ANDREW/AU
L17          90 DUP REM L16 (12 DUPLICATES REMOVED)
L18           2 S L17 AND L1

     FILE 'STNGUIDE' ENTERED AT 11:47:47 ON 17 OCT 2002

     FILE 'BIOSIS, CABA, CAPLUS, EMBASE, LIFESCI, MEDLINE, SCISEARCH,
     USPATFULL, JAPIO' ENTERED AT 11:49:39 ON 17 OCT 2002
L19           5 S KROLL, JOHN SIMON/AU
L20           5 DUP REM L19 (0 DUPLICATES REMOVED)
L21           4 S LANGFORD, PAUL RICHARD/AU

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21 ANSWER 1 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1994:415011 BIOSIS
DN PREV199497428011
TI Molecular aspects of the pathogenesis of invasive Haemophilus influenzae type b infections.
AU Kroll, John Simon; **Langford, Paul Richard**
CS St. Mary's Hosp. Med. Sch., London UK
SO Ellis, R. W. [Editor]; Granoff, D. M. [Editor]. Infectious Disease and Therapy, (1994) Vol. 11, pp. 145-177. Infectious Disease and Therapy; Development and clinical uses of Haemophilus b conjugate vaccines. Publisher: Marcel Dekker, Inc. 270 Madison Avenue, New York, New York 10016, USA.
ISSN: 1043-2981. ISBN: 0-8247-9186-X.
DT Book
LA English

L21 ANSWER 2 OF 4 CAPLUS COPYRIGHT 2002 ACS
AB The invention provides methods of screening commensal and pathogenic bacteria for previously unidentified vaccine antigens, based upon identifying polypeptide antigens that bind to sera raised against commensal bacterial proteins. Also provided are vaccine compositions and methods of preparing vaccine compositions comprising the antigens identified by the screening methods. Antigens and uses thereof are also described.
AN 2002:754696 CAPLUS
TI Pathogenic and commensal vaccine antigens
IN Robinson, Andrew; Gorringe, Andrew Richard; Hudson, Michael John; Bracegirdle, Philippa; West, David McKay; Oliver, Kerry Jane; Kroll, John Simon; **Langford, Paul Richard**
PA Microbiological Research Authority, UK; Imperial College Innovations Limited
SO PCT Int. Appl., 310 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2002077648	A2	20021003	WO 2002-GB1399	20020322
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
PRAI	GB 2001-7219	A	20010322		

L21 ANSWER 3 OF 4 CAPLUS COPYRIGHT 2002 ACS
AB The present invention relates to pharmaceutical compns. comprising Cu,Zn-superoxide dismutase (Cu,Zn-SOD) of the dimeric type, nucleic acid encoding a Cu,Zn-SOD, or antibody to a Cu,Zn-SOD for treating and/or vaccinating against bacterial infection. Also described are methods for isolation of Cu,Zn-SODs and for prepn. of pharmaceutical compns., preferably for providing or eliciting protective immunity to meningococcal infection in an animal.
AN 2000:161457 CAPLUS
DN 132:206934
TI Cu,Zn-Superoxide dismutase or antibody thereto as vaccine against bacterial (including meningococcal) infection
IN Gorringe, Andrew Richard; Kroll, John Simon; **Langford, Paul**

Richard; Robinson, Andrew
PA Microbiological Research Authority, UK; Imperial College of Science, Technology and Medicine
SO PCT Int. Appl., 27 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000012718	A1	20000309	WO 1999-GB2828	19990827
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9956350	A1	20000321	AU 1999-56350	19990827
EP 1108038	A1	20010620	EP 1999-943065	19990827
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002523521	T2	20020730	JP 2000-567704	19990827
PRAI GB 1998-18756	A	19980827		
WO 1999-GB2828	W	19990827		

RE.CNT 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L21 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2002 ACS
AB A review, with 121 refs.
AN 1994:652598 CAPLUS
DN 121:252598
TI Molecular aspects of the pathogenesis of invasive Haemophilus influenzae type b infections
AU Kroll, John Simon; **Langford, Paul Richard**
CS St. Mary's Hospital Medical School, London, UK
SO Infectious Disease and Therapy (1994), 11(DEVELOPMENT AND CLINICAL USES OF HAEMOPHILUS B CONJUGATE VACCINES), 145-77
CODEN: IDTHER; ISSN: 1043-2981
DT Journal; General Review
LA English

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SO Infection and Immunity, (March, 2001) Vol. 69, No. 3, pp. 1561-1567.
print.
ISSN: 0019-9567.
DT Article
LA English
SL English

L15 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1999:395782 BIOSIS
DN PREV199900395782
TI The molecular basis of serum resistance in group B meningococci: Inhibition of membrane attack complex insertion by capsular polysaccharide.
AU Mackinnon, Fiona G. (1); Gulati, Sunita; **Gorringe, Andrew**; Oppermann, Martin; Rice, Peter A.; Ram, Sanjay
CS (1) University of Oxford, Oxford UK
SO Molecular Immunology, (March April, 1999) Vol. 36, No. 4-5, pp. 295.
Meeting Info.: 7th European Meeting on Complement in Human Disease Helsinki, Finland June 17-20, 1999
ISSN: 0161-5890.
DT Conference
LA English

L15 ANSWER 4 OF 4 CAPLUS COPYRIGHT 2002 ACS
AB A review with 30 refs. (c) 1998 Academic Press.
AN 1999:6390 CAPLUS
DN 130:107295
TI Characterization of bacterial surface receptor-ligand interactions
AU Holland, Julie; **Gorringe, Andrew**
CS School of Pharmaceutical Sciences, University of Nottingham, University Park, Nottingham, NG7 2RD, UK
SO Methods in Microbiology (1998), 27(Bacterial Pathogenesis), 215-225
CODEN: MMICEU; ISSN: 0580-9517
PB Academic Press
DT Journal; General Review
LA English
RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

20 ANSWER 1 OF 5 CAPLUS COPYRIGHT 2002 ACS
AB The invention provides methods of screening commensal and pathogenic bacteria for previously unidentified vaccine antigens, based upon identifying polypeptide antigens that bind to sera raised against commensal bacterial proteins. Also provided are vaccine compositions and methods of preparing vaccine compositions comprising the antigens identified by the screening methods. Antigens and uses thereof are also described.
AN 2002:754696 CAPLUS
TI Pathogenic and commensal vaccine antigens
IN Robinson, Andrew; Gorringe, Andrew Richard; Hudson, Michael John; Bracegirdle, Philippa; West, David McKay; Oliver, Kerry Jane; **Kroll, John Simon**; Langford, Paul Richard
PA Microbiological Research Authority, UK; Imperial College Innovations Limited
SO PCT Int. Appl., 310 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002077648	A2	20021003	WO 2002-GB1399	20020322

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI GB 2001-7219 A 20010322

L20 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2002 ACS
AB Methods and compns. for the treatment of microbial infection, and in particular meningococcal disease, comprise a commensal Neisseria or an ext. of a commensal Neisseria. Further methods and compns. comprise commensal Neisseria which express genes from virulent strains of Neisseria and/or heterologous gene products from non-neisserial sources. Such compns. are used in vaccine prepns. for the treatment of microbial infection.
AN 2000:608607 CAPLUS
DN 133:213155
TI Neisserial vaccine compositions and methods
IN Robinson, Andrew; Gorringe, Andrew Richard; Hudson, Michael John; Bracegirdle, Philippa; **Kroll, John Simon**; Cartwright, Keith
PA Microbiological Research Authority, UK; Imperial College School of Science, Technology and Medicine; Public Health Laboratory Service Board
SO PCT Int. Appl., 35 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000050074	A2	20000831	WO 2000-GB624	20000222
WO 2000050074	A3	20001228		

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM,

AZ; BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

EP 1154791 A2 20011121 EP 2000-905182 20000222
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

PRAI GB 1999-4028 A 19990222
GB 1999-22561 A 19990923
WO 2000-GB624 W 20000222

L20 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2002 ACS

AB The present invention relates to pharmaceutical compns. comprising Cu,Zn-superoxide dismutase (Cu,Zn-SOD) of the dimeric type, nucleic acid encoding a Cu,Zn-SOD, or antibody to a Cu,Zn-SOD for treating and/or vaccinating against bacterial infection. Also described are methods for isolation of Cu,Zn-SODs and for prepn. of pharmaceutical compns., preferably for providing or eliciting protective immunity to meningococcal infection in an animal.

AN 2000:161457 CAPLUS

DN 132:206934

TI Cu,Zn-Superoxide dismutase or antibody thereto as vaccine against bacterial (including meningococcal) infection

IN Gorringe, Andrew Richard; **Kroll, John Simon**; Langford, Paul Richard; Robinson, Andrew

PA Microbiological Research Authority, UK; Imperial College of Science, Technology and Medicine

SO PCT Int. Appl., 27 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000012718	A1	20000309	WO 1999-GB2828	19990827
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9956350	A1	20000321	AU 1999-56350	19990827
EP 1108038	A1	20010620	EP 1999-943065	19990827
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002523521	T2	20020730	JP 2000-567704	19990827
PRAI GB 1998-18756	A	19980827		
WO 1999-GB2828	W	19990827		

RE.CNT 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L20 ANSWER 4 OF 5 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

AN 1994:415011 BIOSIS

DN PREV199497428011

TI Molecular aspects of the pathogenesis of invasive Haemophilus influenzae type b infections.

AU **Kroll, John Simon**; Langford, Paul Richard

CS St. Mary's Hosp. Med. Sch., London UK

SO Ellis, R. W. [Editor]; Granoff, D. M. [Editor]. Infectious Disease and Therapy, (1994) Vol. 11, pp. 145-177. Infectious Disease and Therapy; Development and clinical uses of Haemophilus b conjugate vaccines.